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to PHARMASEARCH
NEWS 14 Oct 09 Korean abstracts now included in Derwent World Patents
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NEWS 15 Oct 09 Number of Derwent World Patents Index updates increased
NEWS 16 Oct 15 Calculated properties now in the REGISTRY/ZREGISTRY File

NEWS EXPRESS August 15 CURRENT WINDOWS VERSION IS V6.0c,
CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP),
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=> s clenbuterol/cn
L1 1 CLENBUTEROL/CN

=> d

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2001 ACS

RN 37148-27-9 REGISTRY

CN Benzenemethanol, 4-amino-3,5-dichloro-.alpha.-[[(1,1-
dimethylethyl)amino]methyl]- (9CI) (CA INDEX NAME)

OTHER NAMES:

CN (.+-.)-Clenbuterol

CN (RS)-Clenbuterol

CN 1-(4-Amino-3,5-dichlorophenyl)-2-(tert-butylamino)ethanol

CN **Clenbuterol**

CN dl-Clenbuterol

CN P 5369

CN Planipart

FS 3D CONCORD

DR 50306-01-9

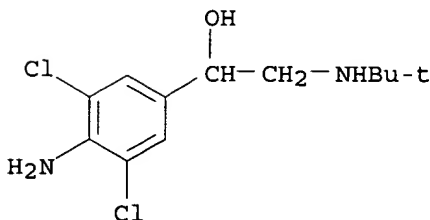
MF C12 H18 Cl2 N2 O

CI COM

LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
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=> fil medlin capl bios embase uspatfull

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BIOSIS - The BIOSIS Previews(R)/RN File 1969-present

ENTER FILE OR CLUSTER NAME (IGNORE):biosis

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FILE 'MEDLINE' ENTERED AT 17:12:28 ON 17 OCT 2001

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=> s l1 or clenbuterol

L2 5083 L1 OR CLENBUTEROL

=> s spinal cord

L3 247825 SPINAL CORD

=> s l2 (s) l3

L4 23 L2 (S) L3

=> dup rem l4

PROCESSING COMPLETED FOR L4

L5 11 DUP REM L4 (12 DUPLICATES REMOVED)

=> d ibib abs kwic 1-5

L5 ANSWER 1 OF 11 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 2001:261570 BIOSIS

DOCUMENT NUMBER: PREV200100261570

TITLE: The therapeutic window for clenbuterol in
spinal cord contusion injury is
severity-dependent.

AUTHOR(S): Zeman, Richard J. (1); Peng, Hong (1); Feng, Yong (1);
Couldwell, William T. (1); Etlinger, Joseph D. (1)

CORPORATE SOURCE: (1) Dept of Cell Biology and Anatomy, New York Medical
College, Valhalla, NY, 10595 USA

SOURCE: FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A1151.
print.

Meeting Info.: Annual Meeting of the Federation of American

DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

AB An important goal of rehabilitation following **spinal cord** injury is recovery of locomotor function and muscular strength. Previously, we found that chronic treatment with the beta2-adrenoceptor agonist, **clenbuterol**, led to enhanced locomotor recovery that was associated with sparing of **spinal cord** tissue following contusion injury. In the present studies, to determine the therapeutic window for **clenbuterol** administration, single injections of **clenbuterol** were administered at timed intervals after injury. A model of **spinal cord** injury was used in which four graded levels of contusion injury were produced in rats with a weight-drop device. A vertical guide allows a 10g rod with 2.5-mm diameter tip to drop from preset heights of either 6.25, 12.5, 25 or 50-mm unto the exposed cord dura at the level of T10. Locomotor function was determined for six weeks following injury with the Basso, Beattie and Bresnahan (BBB) scale. Continuous treatment with **clenbuterol** (1.6 mg/kg/day, drinking H₂O) caused substantial enhancement of recovery of locomotor function at the two most severe levels of injury (BBB scores, 10-12 vs 2-4, $P < 0.05$, LSD). However, an increased dose (16 mg/kg/day) did not improve recovery. Interestingly, single injections of **clenbuterol** (2 mg/kg ip) immediately following injury with a 25-mm weight drop were as effective as chronic treatment. Similar locomotor recovery was observed with injection at 1 day postinjury although attenuation was observed at 2 and 3 days suggesting a time-window of 24 hour postinjury for optimal effectiveness. In contrast to the results with a 25-mm weight drop, single injections of **clenbuterol** immediately following a 50-mm drop did not improve recovery. These results suggest the possibility that additional injury mechanisms are activated by more severe injury, which require a wider therapeutic window in order to achieve recovery of locomotor function.

TI The therapeutic window for **clenbuterol** in **spinal cord** contusion injury is severity-dependent.

AB An important goal of rehabilitation following **spinal cord** injury is recovery of locomotor function and muscular strength. Previously, we found that chronic treatment with the beta2-adrenoceptor agonist, **clenbuterol**, led to enhanced locomotor recovery that was associated with sparing of **spinal cord** tissue following contusion injury. In the present studies, to determine the therapeutic window for **clenbuterol** administration, single injections of **clenbuterol** were administered at timed intervals after injury. A model of **spinal cord** injury was used in which four graded levels of contusion injury were produced in rats with a weight-drop device. A . . . Locomotor function was determined for six weeks following injury with the Basso, Beattie and Bresnahan (BBB) scale. Continuous treatment with **clenbuterol** (1.6 mg/kg/day, drinking H₂O) caused substantial enhancement of recovery of locomotor function at the two most severe levels of injury. . . . scores, 10-12 vs 2-4, $P < 0.05$, LSD). However, an increased dose (16 mg/kg/day) did not improve recovery. Interestingly, single injections of **clenbuterol** (2 mg/kg ip) immediately following injury with a 25-mm weight drop were as effective as chronic treatment. Similar locomotor recovery. . . of 24 hour postinjury for optimal effectiveness. In contrast to the results with a 25-mm weight drop, single injections of **clenbuterol** immediately following a 50-mm drop did not improve recovery. These results suggest the possibility that additional injury mechanisms are activated. . . .

L5 ANSWER 2 OF 11 MEDLINE DUPLICATE 1
 ACCESSION NUMBER: 2001550030 IN-PROCESS
 DOCUMENT NUMBER: 21469729 PubMed ID: 11586111
 TITLE: Acute spinal cord injury, part II: contemporary pharmacotherapy.
 AUTHOR: Dumont R J; Verma S; Okonkwo D O; Hurlbert R J; Boulos P T; Ellegala D B; Dumont A S
 CORPORATE SOURCE: Faculty of Medicine, University of British Columbia, Vancouver, British Columbia, Canada.
 SOURCE: CLINICAL NEUROPHARMACOLOGY, (2001 Sep-Oct) 24 (5) 265-79. Journal code: CNK; 7607910. ISSN: 0362-5664.
 PUB. COUNTRY: United States
 LANGUAGE: English
 FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
 ENTRY DATE: Entered STN: 20011015
 Last Updated on STN: 20011015

AB **Spinal cord** injury (SCI) remains a common and devastating problem of modern society. Through an understanding of underlying pathophysiologic mechanisms involved in the evolution of SCI, treatments aimed at ameliorating neural damage may be developed. The possible pharmacologic treatments for acute **spinal cord** injury are herein reviewed. Myriad treatment modalities, including corticosteroids, 21-aminosteroids, opioid receptor antagonists, gangliosides, thyrotropin-releasing hormone (TRH) and TRH analogs, antioxidants and free radical scavengers, calcium channel blockers, magnesium replacement therapy, sodium channel blockers, N-methyl-D-aspartate receptor antagonists, alpha-amino-3-hydroxy-5-methylisoxazole-4-propionic acid-kainate receptor antagonists, modulators of arachadonic acid metabolism, neurotrophic growth factors, serotonin antagonists, antibodies against inhibitors of axonal regeneration, potassium channel blockers (4-aminopyridine), paclitaxel, **clenbuterol**, progesterone, gabexate mesylate, activated protein C, caspase inhibitors, tacrolimus, antibodies against adhesion molecules, and other immunomodulatory therapy have been studied to date. Although most of these agents have shown promise, only one agent, methylprednisolone, has been shown to provide benefit in large clinical trials. Given these data, many individuals consider methylprednisolone to be the standard of care for the treatment of acute SCI. However, this has not been established definitively, and questions pertaining to methodology have emerged regarding the National Acute **Spinal Cord** Injury Study trials that provided these conclusions. Additionally, the clinical significance (in contrast to statistical significance) of recovery after methylprednisolone treatment is unclear and must be considered in light of the potential adverse effects of such treatment. This first decade of the new millennium, now touted as the Decade of the Spine, will hopefully witness the emergence of universal and efficacious pharmacologic therapy and ultimately a cure for SCI.

AB **Spinal cord** injury (SCI) remains a common and devastating problem of modern society. Through an understanding of underlying pathophysiologic mechanisms involved in the evolution of SCI, treatments aimed at ameliorating neural damage may be developed. The possible pharmacologic treatments for acute **spinal cord** injury are herein reviewed. Myriad treatment modalities, including corticosteroids, 21-aminosteroids, opioid receptor antagonists, gangliosides, thyrotropin-releasing hormone (TRH) and TRH analogs, . . . of arachadonic acid metabolism, neurotrophic growth factors, serotonin antagonists, antibodies against inhibitors of axonal regeneration, potassium channel blockers (4-aminopyridine), paclitaxel, **clenbuterol**, progesterone, gabexate mesylate, activated protein C, caspase inhibitors, tacrolimus, antibodies against adhesion molecules, and other immunomodulatory therapy have been studied. . . acute SCI.

However, this has not been established definitively, and questions pertaining to methodology have emerged regarding the National Acute **Spinal Cord** Injury Study trials that provided these conclusions. Additionally, the clinical significance (in contrast to statistical significance) of recovery after methylprednisolone. . .

L5 ANSWER 3 OF 11 USPATFULL

ACCESSION NUMBER: 2000:7338 USPATFULL

TITLE: Method for treating scoliosis with .beta.2-adrenoceptor agonists

INVENTOR(S): Etlinger, Joseph D., Mt. Kisco, NY, United States
Zeman, Richard J., New York, NY, United States

PATENT ASSIGNEE(S): New York Medical College, Valhalla, NY, United States
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6015837		20000118
APPLICATION INFO.:	US 1997-920018		19970826 (8)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-25147	19960829 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	Granted	
PRIMARY EXAMINER:	Krass, Frederick	
LEGAL REPRESENTATIVE:	Skadden, Arps, Slate, Meagher & Flom LLP, Sommer, Evelyn M.	
NUMBER OF CLAIMS:	12	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	3 Drawing Figure(s); 4 Drawing Page(s)	
LINE COUNT:	573	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of treating scoliosis by administering .beta..sub.2 adrenergic agonists in amounts sufficient to correct the condition.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DRWD FIG. 2. Dorsoventral radiograms of rats showing thoracic scoliosis 4 weeks after three-fourths **spinal cord** transection at T5 (1st panel on the left) or lumbar scoliosis after three-fourths transection at T11 (3rd panel). Radiograms shown in 2nd and 4th panels demonstrate that scoliosis is greatly reduced by **clenbuterol** after three-fourths transection at T5 and T11, respectively.

DETD An animal model of **spinal cord** injury was used to produce neuromuscular scoliosis for treatment with **clenbuterol**. This model uses an asymmetric injury to the **spinal cord** and thus is a valid model for scoliosis, which has been shown to result from a variety of procedures including. . . the dorsal columns or posterior horn (Barrios 1987, Pincott I 1982) and either the posterior or anterior roots of the **spinal cord** (Suk 1989, Pincott II 1984). In the present experiments, rat **spinal cords** were sectioned three-quarters of the way through, beginning on the right side, so that the left lateral columns remained intact. . .

DETD Unexpectedly, significant lateral displacement of the vertebrae was absent in groups of rats that were treated with **clenbuterol** for 4 weeks following identical three-quarter transection of the **spinal cord** at T5 or T11 (FIG. 1). The mechanism for this result is unknown, as was its existence prior to this. . .

L5 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1999:166517 CAPLUS

DOCUMENT NUMBER: 130:205147
 TITLE: Use of .beta.2-adrenoceptor agonists for the treatment of scoliosis
 INVENTOR(S): Etlinger, Joseph D.; Zeman, Richard J.
 PATENT ASSIGNEE(S): New York Medical College, USA
 SOURCE: PCT Int. Appl., 30 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9909966	A1	19990304	WO 1998-US12864	19980618
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6015837	A	20000118	US 1997-920018	19970826
AU 9879820	A1	19990316	AU 1998-79820	19980618
PRIORITY APPLN. INFO.:			US 1997-920018	19970826
			WO 1998-US12864	19980618
AB A method of treating scoliosis by administering .beta.2 adrenergic agonists in amts. sufficient to correct the condition is disclosed. Rat model of spinal cord injury was used to produce neuromuscular scoliosis for treatment with clenbuterol (I). I was added to the drinking water at a dose of 9 mg/L. The successful treatment of scoliosis in rats is reported.				
REFERENCE COUNT: 4				
REFERENCE(S): (1) Alcon Laboratories Inc; WO 8910120 A 1989 CAPLUS (2) Beecham Group PLC; EP 0085514 A 1983 CAPLUS (3) The Rowett Research Institute; EP 0662324 A 1995 CAPLUS (4) Zeman, R; Am J Physiol 1997, V272(4), PE712 CAPLUS				
AB A method of treating scoliosis by administering .beta.2 adrenergic agonists in amts. sufficient to correct the condition is disclosed. Rat model of spinal cord injury was used to produce neuromuscular scoliosis for treatment with clenbuterol (I). I was added to the drinking water at a dose of 9 mg/L. The successful treatment of scoliosis in rats is reported.				
L5 ANSWER 5 OF 11 BIOSIS COPYRIGHT 2001 BIOSIS				
ACCESSION NUMBER: 2000:68469 BIOSIS				
DOCUMENT NUMBER: PREV200000068469				
TITLE: Clenbuterol improves behavioral recovery following chronic spinal cord injury.				
AUTHOR(S): Sayers, S. (1); Lucero, Y.; Mrkvika, R. (1); Khan, N. (1); Chhangani, V. (1); Chauhan, N. (1); Khan, T. (1)				
CORPORATE SOURCE: (1) Research Service, Hines VA Hospital, Hines, IL USA				
SOURCE: Society for Neuroscience Abstracts, (1999) Vol. 25, No. 1-2, pp. 493. Meeting Info.: 29th Annual Meeting of the Society for Neuroscience, Part 1 Miami Beach, Florida, USA October 23-28, 1999 The Society for Neuroscience . ISSN: 0190-5295.				
DOCUMENT TYPE: Conference				
LANGUAGE: English				

TI **Clenbuterol** improves behavioral recovery following chronic
spinal cord injury.

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(Y)/N:y

L5 ANSWER 6 OF 11 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999417619 MEDLINE
DOCUMENT NUMBER: 99417619 PubMed ID: 10486195
TITLE: **Clenbuterol**, a beta(2)-adrenoceptor agonist,
improves locomotor and histological outcomes after
spinal cord contusion in rats.
AUTHOR: Zeman R J; Feng Y; Peng H; Etlinger J D
CORPORATE SOURCE: Department of Cell Biology and Anatomy, New York Medical
College, Valhalla, New York, 10595, USA.
SOURCE: EXPERIMENTAL NEUROLOGY, (1999 Sep) 159 (1) 267-73.
Journal code: EQF; 0370712. ISSN: 0014-4886.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991014
Last Updated on STN: 19991014
Entered Medline: 19991007
AB An important goal of rehabilitation following **spinal**
cord injury is recovery of locomotor function and muscular
strength. In the present studies, we determined whether the
beta(2)-agonist, **clenbuterol**, can improve recovery of locomotor
function following **spinal cord** injury. A model of
spinal cord injury was examined in which four graded
levels of contusion injury were produced in rats at the level of T10 with
a weight-drop device. Locomotor recovery was determined with the Basso,
Beattie, and Bresnahan (BBB) scale, which distinguishes between 22
progressive levels of recovery. As observed previously, recovery during
the 6 weeks following injury was inversely related to the severity of

injury. However, **clenbuterol** caused substantial enhancement of recovery of locomotor function at the two most severe levels of injury (BBB scores 10-12 vs 2-4). In addition, the extent of recovery was directly related to sparing of **spinal cord** tissue at the contusion center in both untreated and **clenbuterol**-treated **spinal cords**. Optimization of beta(2)-agonist treatment may lead to a useful therapeutic modality for treatment of **spinal cord** contusion injury.

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TI **Clenbuterol**, a beta(2)-adrenoceptor agonist, improves locomotor and histological outcomes after **spinal cord** contusion in rats.

AB An important goal of rehabilitation following **spinal cord** injury is recovery of locomotor function and muscular strength. In the present studies, we determined whether the beta(2)-agonist, **clenbuterol**, can improve recovery of locomotor function following **spinal cord** injury. A model of **spinal cord** injury was examined in which four graded levels of contusion injury were produced in rats at the level of T10. . recovery. As observed previously, recovery during the 6 weeks following injury was inversely related to the severity of injury. However, **clenbuterol** caused substantial enhancement of recovery of locomotor function at the two most severe levels of injury (BBB scores 10-12 vs 2-4). In addition, the extent of recovery was directly related to sparing of **spinal cord** tissue at the contusion center in both untreated and **clenbuterol**-treated **spinal cords**. Optimization of beta(2)-agonist treatment may lead to a useful therapeutic modality for treatment of **spinal cord** contusion injury.

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L5 ANSWER 7 OF 11 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 2000:68448 BIOSIS

DOCUMENT NUMBER: PREV200000068448

TITLE: Muscle atrophy reversed by **clenbuterol** following chronic **spinal cord** injury.

AUTHOR(S): Khan, T. (1); Sayers, S. (1); Mrkvicka, R.; Chauhan, N. (1); Hussain, J.; Lucero, Y.

CORPORATE SOURCE: (1) Rehabilitation R and D Section, Hines VA Hospital, Hines, IL USA

SOURCE: Society for Neuroscience Abstracts, (1999) Vol. 25, No. 1-2, pp. 218.

Meeting Info.: 29th Annual Meeting of the Society for Neuroscience, Part 1 Miami Beach, Florida, USA October 23-28, 1999 The Society for Neuroscience . ISSN: 0190-5295.

DOCUMENT TYPE: Conference

LANGUAGE: English

TI Muscle atrophy reversed by **clenbuterol** following chronic **spinal cord** injury.

L5 ANSWER 8 OF 11 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1999:46765 BIOSIS

DOCUMENT NUMBER: PREV199900046765

TITLE: **Clenbuterol** improves behavioral recovery after **spinal cord** injury.

AUTHOR(S): Sayers, S. (1); Lucero, Y.; Mrkvicka, R. (1); Khan, N. (1); Chhangani, V. (1); Chauhan, N. (1); Khan, T. (1)

CORPORATE SOURCE: (1) Res. Serv., Hines V.A. Hosp., Hines, IL 60141 USA

SOURCE: Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp. 309.

Meeting Info.: 28th Annual Meeting of the Society for

Neuroscience, Part 1 Los Angeles, California, USA November
7-12, 1998 Society for Neuroscience
. ISSN: 0190-5295.

DOCUMENT TYPE: Conference

LANGUAGE: English

TI **Clenbuterol** improves behavioral recovery after **spinal
cord** injury.

L5 ANSWER 9 OF 11 MEDLINE DUPLICATE 3

ACCESSION NUMBER: 97287766 MEDLINE

DOCUMENT NUMBER: 97287766 PubMed ID: 9142894

TITLE: **Clenbuterol**, a beta2-adrenoceptor agonist,
reduces scoliosis due to partial transection of rat
spinal cord.

AUTHOR: Zeman R J; Zhang Y; Etlinger J D

CORPORATE SOURCE: Department of Cell Biology and Anatomy, New York Medical
College, Valhalla 10595, USA.

SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1997 Apr) 272 (4 Pt 1)
E712-5.

PUB. COUNTRY: Journal code: 3U8; 0370511. ISSN: 0002-9513.

LANGUAGE: English
Journal; Article; (JOURNAL ARTICLE)

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199706

ENTRY DATE: Entered STN: 19970620
Last Updated on STN: 19970620
Entered Medline: 19970606

AB Injury to the **spinal cord** often results in abnormal
lateral curvature of the spine, or scoliosis, that is associated with
neuromuscular weakness. The lateral curvature of the spine is thought to
be a consequence of insufficient or asymmetrical loading of the vertebrae.
To study neuromuscular scoliosis, an animal model of **spinal
cord** injury was used in which the **spinal cord**
was partially (3/4) transected, with the left lateral columns left intact.
Partial transection of the **spinal cord** in the rat
caused scoliosis that was maximal four to five vertebrae distal to the
lesion site. As in previous experiments involving unilateral
spinal cord lesions, the scoliotic curves were convex on
the weakened side. Subtotal transection at T5 or T11 resulted in lateral
displacement of vertebrae T9-T12 or L2-L5, respectively, of up to 11 mm.
Interestingly, this vertebral displacement is greatly reduced by
clenbuterol, a beta2-adrenoceptor agonist that has been found to
retard loss of muscle contractility and bone mineralization due to
denervation. Together these results suggest that stimulation of
beta2-receptors opposes vertebral unloading due to neuromuscular weakness
and thereby acts as a countermeasure to scoliosis.

TI **Clenbuterol**, a beta2-adrenoceptor agonist, reduces scoliosis due
to partial transection of rat **spinal cord**.

AB Injury to the **spinal cord** often results in abnormal
lateral curvature of the spine, or scoliosis, that is associated with
neuromuscular weakness. The lateral curvature. . . to be a consequence
of insufficient or asymmetrical loading of the vertebrae. To study
neuromuscular scoliosis, an animal model of **spinal cord**
injury was used in which the **spinal cord** was partially
(3/4) transected, with the left lateral columns left intact. Partial
transection of the **spinal cord** in the rat caused
scoliosis that was maximal four to five vertebrae distal to the lesion
site. As in previous experiments involving unilateral **spinal
cord** lesions, the scoliotic curves were convex on the weakened
side. Subtotal transection at T5 or T11 resulted in lateral displacement
of vertebrae T9-T12 or L2-L5, respectively, of up to 11 mm. Interestingly,

this vertebral displacement is greatly reduced by **clenbuterol**, a beta2-adrenoceptor agonist that has been found to retard loss of muscle contractility and bone mineralization due to denervation. Together. . .

L5 ANSWER 10 OF 11 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 91336405 MEDLINE
DOCUMENT NUMBER: 91336405 PubMed ID: 1678583
TITLE: Clenbuterol, a beta 2-receptor agonist, reduces net bone loss in denervated hindlimbs.
AUTHOR: Zeman R J; Hirschman A; Hirschman M L; Guo G; Etlinger J D
CORPORATE SOURCE: Department of Cell Biology and Anatomy, New York Medical College, Valhalla 10595.
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1991 Aug) 261 (2 Pt 1) E285-9.
PUB. COUNTRY: United States
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199109
ENTRY DATE: Entered STN: 19911006
Last Updated on STN: 19950206
Entered Medline: 19910919

AB **Clenbuterol** treatment for several weeks prevented up to one-third of the reduction in mineralization of femurs and tibias caused by sectioning of the sciatic nerve in young rats. The normalizing effect of **clenbuterol** on bone mineral content was directly proportional to similar alterations in muscle mass, which in turn could be abolished by ablation of the triceps surae or hindlimb unweighting and reduced by hypophysectomy. In contrast to the effects of inactivity, ovariectomy caused small reductions (2-4%) in bone density that were not affected by **clenbuterol** and were not accompanied by changes in ash weight. Together, our results suggest that the ability of beta 2-agonists to retard the loss in net muscle mass and enhance contractile tension can oppose net bone loss caused by denervation. Increases in contractile tension caused by beta 2-agonists may enhance the utility of exercise or electrical stimulation as countermeasures for the effects of scoliosis, prolonged bed rest, **spinal cord** injury, or weightlessness in space on bone mass.

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L5 ANSWER 11 OF 11 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 91079204 MEDLINE
DOCUMENT NUMBER: 91079204 PubMed ID: 2258412
TITLE: Determination of clenbuterol in bovine plasma and tissues by gas chromatography-negative-ion chemical ionization mass spectrometry.
AUTHOR: Girault J; Fourtillan J B
CORPORATE SOURCE: CEMAF S.A., Poitiers, France.
SOURCE: JOURNAL OF CHROMATOGRAPHY, (1990 Sep 28) 518 (1) 41-52.

Journal code: HQF; 0427043. ISSN: 0021-9673.
 PUB. COUNTRY: Netherlands
 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199101
 ENTRY DATE: Entered STN: 19910322
 Last Updated on STN: 19910322
 Entered Medline: 19910131

AB A highly sensitive and specific assay was developed for the determination of **clenbuterol** in bovine plasma and tissues. **Clenbuterol** and the internal standard [2H9]**clenbuterol** were measured by gas chromatography-negative-ion chemical ionization mass spectrometry with methane as the reagent gas. Bovine tissues including muscle, liver, heart, kidney, lung, suet, brain, **spinal cord** and thymus were ground in a buffer of pH 7 and then extracted using ethyl acetate. After two subsequent purification steps, the cleaned-up organic extract was derivatized with pentafluoropropionic anhydride. The mass spectrometer was set to monitor the abundant ions m/z 368 and 377 of the perfluoroacyl derivatives. This assay was performed with 1 ml of plasma or 0.2 g of tissue. The feasibility of this method was demonstrated by the determination of **clenbuterol** residues as the femtomole level in a variety of tissues.

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